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The Global Burden of Air Pollution on Mortality: The Need to Include Exposure to Household Biomass Fuel–Derived Particulates

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Anenberg et al. (2010) demonstrated that global mortality associated with outdoor ozone and particulate matter (PM) exposure has been underestimated and that anthropogenic atmospheric PM rather than ozone is the main contributor to death. Although we acknowledge that their investigation was concerned with outdoor air pollution alone, we feel that attention should be drawn to the burden of disease from household air pollution.

Half the world's population is exposed to fine PM [< 2.5 μm in aerodynamic diameter ($PM_{2.5}$)] in their own homes as a consequence of using biomass fuels such as wood, charcoal, and animal/crop residues for cooking, lighting, and heating. Such exposure is prolonged, extensive, and overlooked by examination of atmospheric models alone (Torres-Duque et al. 2008).

Combustion of biomass fuels has been repeatedly demonstrated to produce high concentrations of domestic air pollution, with $PM_{2.5}$ exposures extending in to the milligram per cubic meter range, orders of magnitude above concentrations from exposure to anthropogenic particulate pollution outdoors (Regalado et al. 2006). Rural populations, and women in particular, are likely to have particularly high indoor exposures because of the extended time spent on cooking and household activity (Mestl et al. 2007).

Anenberg et al. (2010) used exposure–response functions derived from epidemiological studies of outdoor air, which emphasize cardiopulmonary mortality in older cohorts. Household air pollution from biomass fuel combustion contributes to chronic respiratory disease and cardiorespiratory events. However, it is particularly implicated in pneumonia in young children (Dherani et al. 2008) and has been ranked the 11th most important risk factor in global mortality, predominantly because of the association with infection (Ezzati et al. 2004). These early deaths would contribute considerably to the estimate of years of life lost due to PM.

We agree with Anenberg et al. (2010) that anthropogenic PM is an important global cause of premature death. However, outdoor levels report only part of the picture and may significantly underestimate the total PM-related mortality burden.

Recent work (Pope et al. 2009) has brought together data on exposure–response functions for outdoor air pollution and cigarette smoking, and there is a need for additional similar work to integrate studies on indoor biomass combustion (Ezzati et al. 2000). These studies would help clarify the exposure–response function of household air pollution as well as assist in the important process of identifying the most cost-efficient means of reducing exposure among the 3 billion people who bear the health burden from high particulate concentrations at home.

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The Global Burden of Air Pollution on Mortality: Anenberg et al. respond

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We appreciate the comments by Rylance et al. stimulated by our analysis of the global burden of disease due to outdoor air pollution (Anenberg et al. 2010). We acknowledge that indoor air pollution is—and has long been recognized as—a significant burden on public health, particularly in developing countries where solid fuels are used extensively for cooking and heating (e.g., Smith 1987; Smith et al. 2004), but these comments on indoor air pollution do not affect our conclusions about the impacts of outdoor air pollution on global mortality.

In the 2004 World Health Organization (WHO) comparative risk assessment, Ezzati et al. (2004) estimated that indoor air pollution associated with household use of solid fuels is responsible for more premature mortalities than outdoor air pollution. Although our estimate of premature mortality due to outdoor air pollution is higher than the previous WHO estimate (Cohen et al. 2004), it should only be compared with indoor air pollution when methods for both risk factors are updated consistently, as in the forthcoming Global Burden of Diseases, Injuries, and Risk Factors Study (Institute for Health Metrics and Evaluation 2010).

We agree with Rylance et al. that the approach used by Pope et al. (2009) to integrate outdoor air pollution and cigarette smoking on a common scale would potentially also be useful for analyzing indoor air pollution. More broadly, additional research is needed to understand and differentiate indoor and outdoor exposures to multiple air pollutants and their ultimate effects on health in different parts of the world.

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A Hybrid Approach for Predicting $PM_{2.5}$ Exposure

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van Donkelaar et al. (2010) integrated the satellite-based aerosol optical depth (AOD) and the chemical transport models (CTM) to develop concentrations of particulate matter < 2.5 μm in aerodynamic diameter ($PM_{2.5}$). Because spatiotemporal coverage of *in situ* air pollution monitoring is limited, the integration of AODs with CTM is the wave of the future for developing time–space (and potentially source) resolved estimates of air quality. However, these methodologies have inherent limitations that the authors failed to address. van Donkelaar et al. (2010) based their research on work of Liu et al. (2004, 2007), but later research from the same authors (Paciorek and Liu 2009) acknowledged the limitations of Liu et al.'s earlier research. van Donkelaar et al. (2010) cited this research but did not address these limitations.

van Donkelaar et al. (2010) conceptualized that $PM_{2.5} = \eta \times AODs$, where η is influenced by relative humidity (≥ 35 and ≥ 50% for North America and Europe, respectively) and computed using AOD_c, the AOD from three-dimensional chemical transport models (3-D CTM). This has several problems: Failing to account for other factors, including boundary layer height, atmospheric pressure, and surface characteristics, can bias $PM_{2.5}$ prediction. van Donkelaar et al. computed η at 2° × 2.5° and then interpolated η at 0.1° × 0.1°, which must have resulted in the same value of η for all 10 km AODs within each 2° × 2.5°area (at the equator), and hence strong spatial autocorrelation in the predicted $PM_{2.5}$. Because the average lifetime of aerosols is one week and aerosols move across geographic space and time, AODs (i.e., the extinction of beam power due to the presence of aerosols) records a very strong spatiotemporal structure. Failing to account for spatiotemporal structure in AODs is likely to produce biased estimates of $PM_{2.5}$ (Kumar 2010).

The CTM is a data-driven methodology, and the robustness of its output is largely dictated by input emission and meteorological data. Because such data are rarely complete and 100% accurate, it is difficult to accurately predict $PM_{2.5}$ and AOD_c using CTM. Researchers are moving toward data assimilation techniques, in which predicted values are calibrated with respect to *in situ* measurements. van Donkelaar et al. failed to take advantage of data assimilation techniques to calibrate AODc.

Because of problems with version 5.0 or earlier of AODs (Levy et al. 2007), NASA is developing a Deep Blue version to estimate AODs over bright surfaces (Hsu 2010). Given the methodological constraints described above, I question van Donkelaar et al.'s (2010) conclusions. In their figures, the predicted $PM_{2.5}$ in sub-Saharan Africa was unexpectedly high. It is unclear how coarse dust in that part of the world could result in high $PM_{2.5}$ concentrations. This must be a result of the overestimated AODs due to surface brightness

The integration of AODs and CTM, coupled with spatiotemporal dynamic modeling, holds great potential to develop time–space resolved estimates of PM. Future research should be geared toward assimilation of the strengths of these methodologies. CTM has a great temporal resolution and is not constrained by cloud cover or biased by surface brightness, but the reliability of CTM output is dictated by the quality of input data. AODs have great spatial resolution (10 km) and can be estimated at finer spatial resolutions (5 km and 2 km), which is likely to be more robust than the coarse resolution AOD (Kumar et al. 2007); however, under cloud-free conditions it captures only two snapshots (at ~ 1030 hours and ~ 1330 hours local overpass time of the Terra and Aqua satellites) per day. Calibrating AODs for the problems mentioned above, daily (morning and afternoon) AODs can be produced globally. The best approach to integrating the strengths of these two methodologies would be to *a*) develop an empirical relationship between the calibrated AODs and AOD_c (estimated using a nested grid at a fine spatial resolution); *b*) utilize this relationship to predict a calibrated AOD_c ($\hat{A}OD_c$) for all data points with available AOD_c; *c*) utilize $\hat{A}OD_c$ to predict $PM_{2.5c}$ concentrations; *d*) develop an empirical relationship between predicted $PM_{2.5c}$ and *in situ* measurements of $PM_{2.5}$ with the adequate control for spatiotemporal structures and other subsidiary variables; and *e*) utilize this empirical relationship to develop the calibrated $\tilde{P}M_{2.5c}$ ($PM_{2.5c}$ predicted using the the empirical model) for all data points for which $PM_{2.5c}$ is available. $\tilde{P}M_{2.5c}$ in turn, can be aggregated and/or interpolated to any spatiotemporal scales using time–space Kriging, an interpolation method that minimizes error in the predicted values across geographic space and time.

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A Hybrid Approach for Predicting $PM_{2.5}$ Exposure: van Donkelaar et al. Respond

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We thank Kumar for his comments on our article (van Donkelaar et al. 2010). We agree that integration of satellite-based aerosol optical depth (AOD) with a chemical transport model (CTM) is valuable to develop estimates of air quality. We also agree that despite the major recent advancements in remote sensing and CTMs, further development of these methods would continue to improve the estimates of fine particulate matter [< 2.5 μm in aerodynamic diameter ($PM_{2.5}$)]. We are grateful for the opportunity to expand on those issues here.

As pointed out by Kumar, the relationship between ground-level $PM_{2.5}$ and AOD is complex, with dependence on the scattering properties of the local aerosol (a function of aerosol type and atmospheric conditions) and their vertical distribution (a function of boundary layer height, transport, production, and loss). These factors include effects of atmospheric pressure and surface concentration. The method we used in our study (van Donkelaar et al. 2010) was designed specifically to account for all of these factors (not only relative humidity, as implied by Kumar). η is defined as the ratio of surface $PM_{2.5}$ to total column AOD, where the definition of $PM_{2.5}$ is at either 35% or 50% relative humidity, in accordance with regional ground measurement standards, and total column AOD includes the effects of local relative humidity on aerosol extinction.

We agree that higher resolution calculations of η would continue to improve the $PM_{2.5}$ estimates and are actively developing this capability. However, it is worth clarifying that the long (~ 1 week) aerosol lifetime does not detract from, but rather it contributes to the accuracy of a simulation at 2° × 2.5°. Short-lived species (< 1 day) have more subgrid spatial variation due to the effects of more rapid atmospheric losses. The smoothing associated with longer-lived aerosols enables a global model to sufficiently capture major processes affecting η.

A number of promising developments are also occurring in satellite remote sensing. The Deep Blue algorithm (Hsu et al. 2006) noted by Kumar is one that attempts to retrieve AOD from MODIS (Moderate Resolution Imaging Spectroradiometer) observations over bright surfaces. We took a different approach by using AOD retrievals from the MISR (Multiangle Imaging Spectroradiometer) instrument, which are robust to surface brightness, and by removing biased AOD retrievals from both MODIS and MISR. We found little bias (< 20%) between AERONET (AErosol RObotic NETwork) and our combined satellite AOD in sub-Saharan Africa. Although our $PM_{2.5}$ estimates over sub-Saharan Africa (van Donkelaar et al. 2010) are subject to uncertainty, recent $PM_{2.5}$ measurements in Ghana (Dionisio et al. 2010) indicate that Saharan dust is a significant regional source of $PM_{2.5}$ and that our estimates may in fact be too low, both in contrast with Kumar's expectations. We welcome additional *in situ* measurements for future comparisons.

The combination of satellite observations and CTMs offers great potential. The approach we presented in our article (van Donkelaar et al. 2010) took advantage of the fine resolution and observational nature of satellite AOD retrievals and estimates ground-level $PM_{2.5}$ using the physically based framework of a CTM. Empirical methods, such as proposed by Kumar, can be effective over regions where sufficient surface measurements are available to train empirical (or semi-empirical) models. However, sufficient *in situ* measurements do not exist for most of the world, thus limiting the geographic scope of any method that is too dependent upon them. Expansion of the current global ground-based aerosol measurement network would provide a valuable data set to evaluate and improve the ability of CTMs to capture the AOD–$PM_{2.5}$ relationship as well as the quality of the resultant satellite-based $PM_{2.5}$ estimate.

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Errata

In the article by Ferreira et al. [Environ Health Perspect 118:249–252 (2010)], the units for fiber length were incorrect in the first column of page 252; "millimeters" should have been "micrometers." The corrected sentence is as follows:

Human macrophages can phagocytose fibers ≤ 20 μm (Zeidler-Erdely et al. 2006).

The authors apologize for the error.

Scinicariello et al. have reported two text errors in their article "Modification by ALAD of the Association between Blood Lead and Blood Pressure in the U.S. Population: Results from the Third National Health and Nutrition Examination Survey" [Environ Health Perspect 118:259–264 (2010)].

First, in the third paragraph of their article (p. 259), the sentence summarizing results of a study of Korean lead smelter workers should have been as follows:

A study conducted among Korean lead smelter workers (n = 798; mean BLL = 32.0 μg/dL) found that the *ALAD* polymorphism did not change the association between blood lead and hypertension at occupational exposure levels compared with *ALAD1* homozygous carriers (Lee et al. 2001).

Second, the sentence in the third paragraph of the "Discussion" (p. 262) was incorrect. The corrected sentence is as follows:

Two previous studies on *ALAD*, BLL, and BP—one conducted among occupationally exposed workers (Lee et al. 2001) and the other conducted at lower lead exposure level (Smith et al. 1995)—found no association of *ALAD* polymorphism and BP outcomes.

The authors apologize for the errors.